Pituitary gland diseases

Learning objectives

Structure and function of pituitary gland parts-1

Causes of anterior pituitary hormones deficiency-2

Clinical presentations of anterior pituitary hormones deficiency-3

Important tests used to evaluate deficiency-4

Management of patient with prove hormone deficiency-5

Ant. Pit. Hormones ⊐ ACTH, TSH, LH and FSH, G.H and prolactin

Post pit. Hormones ⊐ ADH, oxytocin

C/F of pituitary gland disease:

1-Incidental.

2-Effect ⊐ Amenorrhoea or hyper prolactinaemia.

3-Mechanical effect ⊐ visual field defect.

Ant. Pit. Hormone deficiency: 'Hypopituitarism" lead to combined deficiency of any of the ant. Pit. Hormones.

Causes:

1-Structural:-* pit. Tumours (adenoma or rarely carcinoma).

*Secondary tumour (including) leuk. and lymphoma.

*Carniopharyngioma.

*Meningioma.

*Haemorrhage.

2-Inflammatory/ infiltrative:- *sarcoidosis.

* Lymphocytic hypophysitis.

* Infection:- T.B. syphilitis, encephalitis.

*Haemochromatosis.

3-Congenital deficiencies: *GnRH (Kallmmans syndrome).

TRH - CRH

4- Functional:- *chronic systemic illness.

* Anorexia nervosa.

*Excessive exercise.

5- Other:

*Head injury.

*Sellar surgery.

*Sellar radiotherapy.

*Post – partum necrosis.

C/F. depend on:

A- The underlying lesion.

B- Pattern of resulting hormone deficiency:

1- G.H secretion is often the earliest to be lost (lethargy, muscle weakness, and fat mass).

2- Next, gonadotrophin (LH, FSH) secretion becomes impaired (loss of libido in male and oligomenorrhoea or amenorrhoea in female). With gynecomastia and decrease frequency of shaving in male. In both sexes axillary and pubic hair becomes sparse or even abscent.

3- ACTH is the next hormone to be lost leading to symptoms of cortisol deficiency However, Angiotensin II-dependent zona glomerulosa function is not lost and so aldosterone secretion maintain normal plasma K^+.

However there may be postural hypotension and dilutional hyponatraemia because:

A- Failure of vasoconstriction in the absence of cortisol resulting in pooling of blood in legs on standing.

B- Increase ADH due to hypotension and cortisol deficiency.

C -Cortisol is required for normal water excretion by the kidney.

In contrast to pigmentation of Addison's disease a degree of pallor is usually present (because of lack of stimulation of melanocytes by B- lipotrophic hormone which is a fragment of the ACTH precursor- peptide).

4- Finally, TSH secretion is lost leading to secondary hypothyroidism, this contribut further to lethargy and cold intolerance. In contrast to primary hypoth; frank myxoedema is rare, because the thyroid gland retains some autonomous function.

The above symptoms is insidious. However, patients sometimes present acutely which precipitated by a mild infection or injury or secondary to pit. Apoplexy.

Investigations:

1- ACTH:

A- Short ACTH stimulation test (250 Mg ACTH "synacthen" by "i.m injection" blood taken at 0, 30 mimits for cortisol and at 0 minutes for ACTH on ice, normal plasma cortisol > 460 nmol/ L (~ 170 Mg/dL).

B- Insulin tolerance test (0.15 u/kg insulin I.V ▭ blood sample 0, 30, 45, 60, 90, 120 for blood sugar and cortisol, when blood glucose < 40 mg/dL ▭ blood cortisol > 550 nmol/L.

2- LH, FSH;

* Male: testesteron, LH, FSH.

* Pre-menopausal ▭ regular mense.

•Post menopausal ▭ LH, FSH > 30 mu/L.

3 -TSH:

* Measure serum thyroxin (TSH often detectable in secondary hypothyroid – due to inactive TSH isoforms in the blood).

4-GH:

Measure immediately after exercise.

Other stimulatory tests (1 hours after going to sleep, frequent sampling during sleep, post exercise, insulin – induced hypoglycemia).

5- Cranial diabetus insipidus (only investigate if patient complain of polyuria, polydipsia which may be masked by ACTH or TSH deficiency).

* Exclude other causes of polyuria ⎵ by blood sugar, K^+, Ca^+ measurements.

* Water deprivation test (No coffee, tea or smoking on the test day, stop intake fluid at 0730 hours), at 0830 body weight, plasma and urine osmolality recorded every 2 hours, for 8 hours, stop if patient lost 3% of body w.t

- if plasma osmolality reach > 300 mos/Kg and urine osm < 600 mos/Kg, then administer DDAVP 2Mg im.)

- In psychogenic polydipsia low plasma osmolality seen at the start of test.

* 5% saline infusion test ⇨ then measure ADH in response to plasma osmolality.

Hormone excess:

1- Random prolactin.

2- Investigate for acromegaly (glucose tolerance test) or cushing syndrome ⎵ if there is clinical features.

3- Visual field testing. MRI, CT.

Management:

1- Treatment of acutely ill patient is similar to that for adrenocotical insufficiency except that sodium depletion is not an important component to correct.

2- Hormone replacement therapy.

3-Treatment of pituitary macroadenoma. (surgical)

Cortisol replacement: H.C but not mineralocational.

Thyroid replacement: 100-150 Mg/day (the aim is to maintain TSH in upper normal limit).

TSH is not helpful in asses response. It's dangerous to give thyroid hormone in patient with adrenocortical insufficiency (crisis).

Sex hormone replacement (for men at any age and for women < 50 years to prevent osteoporosis).

G.H: daily subcutan, for young patient with GH deficiency, renal failure,turner syndrome.